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16. ABSTRACT

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with specific chemicals or compounds. The Office of Emergency and Remedial Response (Superfund) uses these documents in preparing cost-benefit analyses under Executive Order 12991 for decision-making under CERCLA. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data are available. The interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed. Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, RfD_S or subchronic reference dose, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval. The RfD is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan. For compounds for which there is sufficient evidence of carcinogenicity, q_1^*s have been computed, if appropriate, based on oral and inhalation data if available.

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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with ethyl chloride. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary reflecting limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the TOXLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to May, 1986. Secondary sources of information have also been relied upon in the preparation of this report and represent large scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) sources have been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria for Chlorinated Ethanes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-029. NTIS PB81-117400.

U.S. EPA. 1980b. Hazard Profile for Chloroethane. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

U.S. EPA. 1986a. Summary Review of Health Effects Associated with Monochloroethane: Health Issue Assessment. Internal Review Draft. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Air Quality Planning and Standards, Washington, DC.

The intent in these assessments is to suggest acceptable exposure levels for noncarcinogens and risk cancer potency estimates for carcinogens whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard or risk associated with exposure to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, RfD_S (formerly AIS) or subchronic reference dose, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan).

This type of exposure estimate has not been extensively used, or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for RfD_S estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure. These values are developed for both inhalation (RfD_{SI}) and oral (RfD_{SO}) exposures.

The RfD (formerly AIC) is similar in concept and addresses chronic exposure. It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980c) for a discussion of this concept]. The RfD is route-specific and estimates acceptable exposure for either oral (RfD_O) or inhalation (RfD_I) with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for identifying reportable quantities and the methodology for their development is explained in U.S. EPA (1983).

For compounds for which there is sufficient evidence of carcinogenicity RfD_S and RfD values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980c). Since cancer is a process that is not characterized by a threshold, <u>any</u> exposure contributes an increment of risk. For carcinogens, q_1^*s have been computed, if appropriate, based on oral and inhalation data if available.

ABSTRACT

Limited subchronic toxicity testing of ethyl chloride administered by inhalation defines only free-standing NOELs. There are no carcinogenicity or chronic toxicity data on ethyl chloride, although the NTP (1986) is currently completing a chronic bioassay. Risk assessment for carcinogenic or chronic toxicity effects may be performed on the results of this bioassay. The liver, CNS and heart appear to be target organs.

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LIST OF ABBREVIATIONS

bw	Body weight
CNS	Central nervous system
NOAEL	No-observed-adverse-effect-level
NOEL	No-observed-effect level
PEL	Permissible exposure limit
ppm	Parts per million
RfD	Reference dose
RfD_S	Subchronic reference dose
STEL	Short-term exposure level
TLV	Threshold limit value
TWA	Time-weighted average

1. ENVIRONMENTAL CHEMISTRY AND FATE

Selected physical and chemical properties and environmental fate of ethyl chloride are listed in Table 1-1.

In the atmosphere, oxidation of ethyl chloride by reaction with HO radical is expected to be the dominant degradation process. Based on rate constants ranging from 3.9×10^{-13} to 4.4×10^{-13} cm^3/molecules-sec and an ambient hydroxyl concentration of 8.0×10^5 molecules/cm^3 (Atkinson et al., 1979; Butler et al., 1978), the hydroxyl reaction half-life has been calculated to be 23-26 days. Because of its relatively long lifetime in the atmosphere, ethyl chloride would undergo significant aerial transport. Because of its relatively moderate water solubility, wet or dry deposition may also be important in the removal of this compound from the atmosphere.

In water systems, volatilization is likely to be the most significant fate-determining process of ethyl chloride. Dilling (1977) estimated the average half-life for volatilization of 1 ppm ethyl chloride in solution 6.5 cm deep to be 23.1 minutes when stirred at 200 rpm in still air at 25°C. Hydrolysis may also be an important removal process. Mabey and Mill (1978) estimated the half-life of ethyl chloride in aqueous solution at 25°C and pH 7 to be 38 days. Bioaccumulation in aquatic organisms and adsorption to sediments should not be significant.

The half-life of ethyl chloride in soil could not be located in the literature searched. Based on its measured vapor pressure of 755 mm Hg at 20°C (Mackay and Shiu, 1981), ethyl chloride should volatilize rapidly from wet and dry soil surfaces. Residual ethyl chloride in soil that escapes losses from physical and chemical processes should be highly mobile in soil and may leach into groundwater.

TABLE 1-1

Selected Physical and Chemical Properties and
Environmental Fate of Ethyl Chloride

Property	Value	Reference
CAS number:	75-00-3	
Chemical class:	halogenated aliphatic hydrocarbon	
Molecular weight:	64.52	
Vapor pressure:	1000 mm Hg at 20°C	Verschueren, 1983
Water solubility:	5710 mg/ℓ at 20°C	Mackay and Shiu, 1981
Log octanol/water partition coefficient:	1.43	Hansch and Leo, 1985
Bioconcentration factor:	5-7 (estimated)	Lyman et al., 1982
Soil adsorption coefficient:	33-143 (estimated)	Lyman et al., 1982
Half-life:		
Air	23-26 days	Atkinson et al., 1979; Butler et al., 1978 Dilling, 1977
Water	~ hours	
Soil	NA	

NA = Not available

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

2.1. ORAL

Quantitative data regarding the rate or extent of absorption of ethyl chloride by oral administration could not be located in the available literature. Sax (1984) reported that ethyl chloride is rapidly absorbed into the body after oral intake.

2.2. INHALATION

Sax (1984) and Konietzko (1984) reported that ethyl chloride is rapidly absorbed after inhalation.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. HUMAN

Epidemiological data on the effects of ethyl chloride exposure by any route of administration could not be located in the available literature. Lehmann and Flury (1943) reported that acute exposure to ethyl chloride vapors has narcotic effects in man. Twelve-minute exposure to 19,000 ppm (50 g/m^3) caused weak analgesia; stupor, eye irritation and stomach cramps accompanied two inhalations of 40,000 ppm (106 g/m^3).

3.2. ANIMALS

3.2.1. Oral. Data from animal studies are limited to effects from subchronic and acute exposures. Adams et al. (1939) found no adverse effects of 0.5 or 1.0 g/kg ethyl chloride given to an unspecified number of rabbits as 60 oral doses. No further details were provided. Assuming the rabbits were dosed 5 times/day over a 12-day period and weighed 2.0 kg, a NOAEL of 700 mg/kg/day can be calculated.

3.2.2. Inhalation. NTP (1986) is currently concluding toxicity and cancer chronic inhalation bioassays using rats and mice. Relevant toxicity data that would be useful in quantitative risk assessment may result from this study.

Adams et al. (1939) found no effects in rats or rabbits during or after inhalation exposure to 25.4 mg/ℓ (25.4 g/m^3) ethyl chloride, 7.5-8.0 hours/day, 5 days/week for 6.5 months. Endpoints included weekly weights, gross and microscopic examination, and ophthalmoscopic examination (rabbits only). Assuming a rat weighs 0.35 kg and inhales 0.223 mg/m^3/day (U.S. EPA, 1980c), the concentration used corresponds to a NOAEL of ~3.73 g/kg/day.

A report from the Russian literature, however, indicates that adverse effects occur at much lower levels. Troshina (1966) exposed rats to 0.57 mg/ℓ (570 mg/m^3) 4 hours/day for 6 months and reported "changes in liver

function," fatty liver changes, dystrophic changes in the lungs, reduced arterial blood pressure and depressed leukocyte phagocytic activity.

The NTP (1981) found that rats and mice exposed to 2500-19,000 ppm (~6.6-50 g/m³) ethyl chloride daily for 13 weeks showed no treatment-related histopathological effects. This study, reviewed by Landry et al. (1982), was not available for further evaluation.

Rats of both sexes and male dogs exposed to 1600, 4000 or 10,000 ppm (~4.2, 11 or 26 g/m³) ethyl chloride, 6 hours/day, 5 days/week for 2 weeks showed no treatment-related effects on body weights, clinical chemistry, hematology, urinalysis measures, neurology (dogs only), or gross or microscopic pathology (Landry et al., 1982). At 4000 and 10,000 ppm, male rats had slightly increased liver-to-body weight ratios. The 10,000 ppm concentration was associated with a slight lethargy in rats and initial excitability in dogs, both characteristic of an anesthetic effect. Because of the brevity of the treatment duration, quantitative risk assessment for effects from subchronic exposures cannot be performed on these data. In a comparison of the effects of a brief exposure to ethyl chloride on the liver sulfhydryl concentration in male rats and male mice, it was observed that a single 6-hour exposure to 4000 ppm (11 g/m³) depleted hepatic nonprotein sulfhydryl concentrations in both rats and mice (Landry et al., 1982).

Sayers et al. (1929) found that 2% ethyl chloride (~53 g/m³) for 9 hours was associated with histological changes in the liver and kidneys of guinea pigs, and concentrations >15.3% (~404 g/m³) caused death in <1 hour. In an unspecified species, Doering (1975) observed that overdoses of ethyl chloride used as an anesthetic can produce contractile cardiac failure. Torkelson and Rowe (1981) reported that the most serious problems after acute exposure to ethyl chloride, apart from an anesthetic effect, was potentiation of epinepherine-induced cardiac abnormalities.

No evidence for developmental or reproductive toxicity has been reported for ethyl chloride to date.

4. CARCINOGENICITY

4.1. HUMAN AND ANIMAL DATA

Pertinent data regarding the carcinogenic potency of ethyl chloride in humans or experimental animals, by any route of exposure, could not be located in the available literature. The NTP (1986) completed a 2-year bioassay of ethyl chloride using rats and mice exposed by inhalation; chronic quality assessment is currently in progress.

4.2. OTHER RELEVANT DATA

Ethyl chloride in the vapor phase was mutagenic to four strains of Salmonella typhimurium, both with and without metabolic activation (Riccio et al., 1983). It was, however, negative in a Balb/c-3T3 cell transformation assay (Tu et al., 1985).

4.3. WEIGHT OF EVIDENCE

Because there are no available cancer data on ethyl chloride, the compound should be classified in IARC Group 3, or U.S. EPA Group D (U.S. EPA, 1986c). These categories are reserved for chemicals with inadequate evidence for evaluation of human carcinogenicity. Ethyl chloride should be reclassified when the results from the NTP (1986) bioassay become available.

5. REGULATORY STANDARDS AND CRITERIA

The U.S. EPA (1980a) did not derive an ambient water quality criteria for ethyl chloride because of the lack of sufficient mammalian toxicological data. Based on the limited available data, the Agency stated that it was one of the least toxic of the chloroethanes.

The ACGIH (1985) adopted a TLV-TWA for 8-hour exposure to ethyl chloride of 1000 ppm (~2600 mg/m³). In its documentation of the TLV-TWA, the ACGIH (1986) cited acute studies (Lehmann and Flury, 1943) in humans showing weak analgesia at 19,000 ppm and slight symptoms of toxicity at 13,000 ppm. The committee decided to delete a former STEL of 1250 ppm (~3250 mg/m³) until better toxicological and industrial hygiene data become available.

The OSHA PEL (OSHA, 1985) is 1000 ppm for an 8-hour exposure.

6. RECOMMENDATIONS

The two subchronic studies of ethyl chloride exposure (Adams et al., 1939; NTP, 1981) define only free-standing NOELs, and are therefore of limited value in the calculated RfD_S values. The significance of slight hepatic enlargement and narcosis in rats exposed for 2 weeks at lower concentrations (Landry et al., 1982) is unclear. No other available data are useful for quantitative risk assessment.

Estimation of carcinogenic potency should be made if the results of the NTP bioassay are positive in either of the two tested species. Because the U.S. EPA (1986b) based risk assessment on the findings that methyl chloride produced kidney tumors in mice, the issue of the carcinogenicity of the related ethyl chloride is a cause for concern.

If the results of the NTP bioassay are not sufficient for estimation of carcinogenic potential, a threshold toxicity-based RfD may be estimated. The data reviewed in this document suggest that the liver, the CNS and possibly the heart may be target organs for the toxicity of ethyl chloride. The high vapor pressure of this compound (~1000 mm Hg at 20°C) reflects its volatility and suggests that inhalation exposure is probably more likely than oral exposure.

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